WHAT IS CLAIMED IS:

Sub R1

1. ~~A method of treating hypertrophy in a cardiomyocyte cell comprising the step of inhibiting the function of NF-AT3.~~

2. The method of claim 1, wherein inhibiting the function of NF-AT3 comprises inhibiting the dephosphorlyation of NF-AT3.

3. The method of claim 1, wherein inhibiting the function of NF-AT3 comprises reducing the expression of NF-AT3.

Sub R2

4. ~~The method of claim 1, wherein inhibiting the function of NF-AT3 comprises contacting NF-AT3 with an agent that binds to and inactivates NF-AT3.~~

5. The method of claim 1, wherein said method further comprises inhibiting the upregulation of a gene regulated by NF-AT3, wherein said gene is selected from the group consisting of an atrial natriuretic factor gene, a β-myosin heavy chain gene, a β-type natriuretic peptide and an α-skeletal actin gene.

6. The method of claim 1, wherein inhibiting the function of a NF-AT3 comprises inhibiting the interaction of NF-AT3 with GATA4.

7. The method of claim 2, wherein the agent that inhibits dephosphorylation is Cyclosporin A or FK506.

8. The method of claim 3, wherein the agent that reduces the expression of NF-AT3 is an antisense construct.

9. The method of claim 4, wherein the agent that binds to and inactivates NF-AT3 is an antibody preparation or a small molecule inhibitor.

10. The method of claim 9, wherein the antibody preparation comprises a single chain antibody.

11. The method of claim 9, wherein said antibody preparation consists essentially of a monoclonal antibody.

12. The method of claim 5, wherein the agent that inhibits the function of said genes is an antisense construct.

13. A transgenic, non-human mammal, the cells of which comprise a heterologous NF-AT3 gene under the control of a promoter active in eukaryotic cells.

14. The transgenic mammal of claim 13, wherein said mammal is a mouse.

15. The transgenic mammal of claim 13, wherein said heterologous NF-AT3 gene contains at least one mutation that destroys a phosphorylation site.

16. The transgenic mammal of claim 13, wherein said heterologous NF-AT3 gene is human.

17. The transgenic mammal of claim 15, wherein said NF-AT3 gene encodes a protein that lacks one or more phosphorylation sites of wild-type NF-AT3.

18. The transgenic mammal of claim 15, wherein said NF-AT3 gene encodes a protein that lacks all the phosphorylation sites of wild-type NF-AT3.

19. The transgenic mammal of claim 15, wherein said NF-AT3 gene encodes a protein that lacks amino acids 1-137 of wild-type NF-AT3.

20. The transgenic mammal of claim 13, wherein said promoter is a tissue specific promoter.

21. The transgenic mammal of claim 20, wherein said tissue specific promoter is a cardiomyocyte specific promoter.

22. The transgenic mammal of claim 21, wherein said cardiomyocyte specific promoter selected from the group consisting of BNP, β-MHC, cardiac troponin I, α-MHC, SM22α, and α-skeletal actin promoter.

23. A method for screening modulators of cardiac hypertrophy comprising the steps of:

(a) providing a cell having a mutant NF-AT3 gene lacking one or more phosphorylation sites;
(b) contacting said cell with a candidate modulator; and
(c) monitoring said cell for an effect that is not present when said cell is not treated with said candidate modulator.

24. The method of claim 23, wherein said cell is derived from a cardiomyocyte cell line.

25. The method of claim 23, wherein said cell is derived from a primary cardiomyocyte.

26. The method of claim 23, wherein contacting is performed *in vitro*.

27. The method of claim 26, wherein said monitoring comprises measuring the activity or expression of a gene selected from the group consisting of an atrial natriuretic factor gene, a β-myosin heavy chain gene, a cardiac actin gene and an α-skeletal actin gene.

28. The method of claim 24, wherein said monitoring comprises measuring the size or mass of said cell.

29. The method of claim 24, wherein said monitoring comprises monitoring Ca^{++} response in said cell.

30. The method of claim 29, wherein monitoring said Ca^{++} response comprises monitoring Ca^{++} dependent gene expression in said cell.

31. The method of claim 23, wherein said contacting is performed *in vivo*.

32. The method of claim 31, wherein said cell is part of a transgenic, non-human mammal.

33. The method of claim 31, wherein said monitoring comprises measuring cardiac hypertrophy.

34. The method of claim 23, wherein said NF-AT3 gene encodes a protein that lacks one or more phosphorylation sites of wild-type NF-AT3.

35. The method of claim 23, wherein said NF-AT3 gene encodes a protein that lacks all the phosphorylation sites of wild-type NF-AT3.

36. The method of claim 23, wherein said NF-AT3 gene encodes a protein that lacks amino acids 1-137 of wild-type NF-AT3.

37. The method of claim 23, wherein said candidate modulator is an antisense construct.

38. The method of claim 23, wherein said candidate modulator is from a small molecule library.

39. The method of claim 23, wherein said candidate modulator is an antibody.

40. The method of claim 41, wherein said antibody is a single chain antibody.